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ORIGINAL PAPER

Maternal bariatric surgery: adverse outcomes in neonates

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Abstract
Background The obesity epidemic in developed countries has led to an increased prevalence of obese women of reproductive age. As maternal obesity has far-reaching consequences for both mother and child, the consensus is that weight loss before pregnancy will reduce obesity-related morbidity and mortality. Therefore, an increasing number of women become pregnant after undergoing obesity surgery.
Results and discussion From the literature, data shows that perinatal outcome after bariatric surgery is generally considered as favourable for both mother and child. Only a few case reports highlight the possibility of side effects on the foetus and neonate. We report on five cases with severe intracranial bleeding, all possibly related to vitamin K deficiency following maternal bariatric surgery.
Conclusion These reports indicate that careful nutritional follow-up during pregnancy after obesity surgery is mandatory, because nutritional deficiencies such as vitamin K deficiency can lead to life-threatening bleeding.

Keywords Bariatric surgery · Neonatal intracranial bleeding · Obesity · Vitamin K

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Introduction

In Western society, obesity is a major health problem. The World Health Organisation suggests that, by 2015, approximately 2.3 billion adults will be overweight and more than 700 million will be obese [28]. Together with this global epidemic of obesity, there has been a dramatic increase of morbid obesity in women of childbearing age. Obesity in pregnancy represents a special problem, not only because of the adverse effects on maternal health and pregnancy outcome but because the disturbed nutritional balance in utero may even have lifelong deleterious effects on the developing child.

In obese women of reproductive age, it is therefore strongly recommended to lose weight before pregnancy [1]. When behavioural and medical interventions fail, bariatric surgery may be a successful alternative. In general, it is safely recommended to obese women of childbearing age. Weight reduction after surgery improves fertility, results in a significantly lower incidence of severe pregnancy-related complications like hypertension, large-for-gestational-age infants, gestational diabetes, instrumental delivery and

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caesarean section [2, 6, 7, 13–22, 26, 27]. Nevertheless, increased incidences of miscarriage, growth restriction and prematurity have been reported; there are a few case reports and small series that have reported on nutritional deficiencies and related maternal and foetal complications, especially after the malabsorptive types of surgery [4, 12, 23]. Despite the increased incidence of maternal vitamin K deficiency and considering the already very limited placental transfer of vitamin K during normal pregnancy, one might expect an increased vulnerability to vitamin K-deficient bleeding disorders in the offspring of these mothers.

Our perinatal unit recently reported on a case of lethal foetal intracranial bleeding due to vitamin K deficiency after maternal bariatric surgery [25]. We hereby describe four additional cases of life-threatening neonatal bleeding disorders, thereby highlighting the specific characteristics of vitamin K metabolism in pregnancy and the inherent risk of severe deficiency secondary to maternal malnutrition.

Case reports

Case 1

Patient 1 was born at the gestational age (GA) of 31 weeks. His mother, a 29-year-old primigravida, underwent laparoscopic gastric banding 2 years prior to this pregnancy for morbid obesity (137 kg, BMI 47.4 kg/m^2). With this treatment, she lost 21 kg, ending with a BMI of 40.0 kg/m^2 and a pre-pregnancy weight of 116 kg. The gastric band was deflated 2 months before the pregnancy. The patient never had any dietary counselling.

At 17 weeks of gestation, the patient started vomiting and having diarrhoea. Within 14 weeks, there was a weight loss of 19 kg. At 28 weeks, hospital admission was required because of persistent vomiting and the inability to ingest solid foods and liquids. On gastroscopy, a proximal dilatation of the pouch and reflux oesophagitis grade C was diagnosed. This was managed conservatively with intravenous fluids and supplements of water-soluble vitamins (Soluvit Novum®, Fresenius). Three weeks later, maternal vomiting still persisted and the patient was referred to a tertiary centre at a gestational age of 30 weeks. Total parenteral nutrition with water- and fat-soluble vitamins and trace elements was started 1 day before delivery. Initial foetal assessment by ultrasound showed normal foetal growth and normal Doppler. One day after admission, foetal monitoring revealed alternating bradycardia and a sinusoidal pattern and the baby was delivered by emergency caesarean section.

The Apgar score after 1 min was 4 and the baby was immediately intubated and ventilated due to apnoea and severe hypotonia. The growth parameters of the neonate was 2,095 g (P90), length 46.7 cm (P97) and head circumference 34.0 cm (>P97). Clinical examination showed a bulging anterior fontanel and widened cranial sutures suggestive for intracranial haemorrhage.

The haemoglobin level on cord blood was 9.1 g/dL and normal thrombocyte level in the blood: 175×10^9/L. The coagulation values, including the vitamin K-dependent coagulation factors, were severely disturbed (Table 1).

A computer tomography (CT) scan showed a bilateral subdural haematoma of both hemispheres, subarachnoidal haemorrhage, bilateral intraventricular haemorrhage (IVH), cerebral oedema and signs of intracranial hypertension.

He was treated with vitamin K and fresh frozen plasma. Bleeding stopped and the coagulation values normalised from day 3 on.

The baby remained hypotonic and unresponsive and EEGs on days3 and 4 showed a flattened and non-reactive trace. There was no clinical improvement in the next days and in agreement with the parents, intensive care was withdrawn. The baby died on day 5.

The mother's clotting profile showed a prolonged prothrombin time (PT) of 14.7 s and a normal activated partial thromboplastin time (aPTT) of 29 s. Vitamin K-dependent clotting factors were clearly deficient (Table 1). Vitamin K level was 0.2 nmol/L (normal range 0.8–5.3 nmol/L). She also had a low serum protein level (57 g/L) and low vitamin A level (72 μg/L).

After delivery, she continued vomiting and an oesophagogastroduodenoscopy and contrast studies showed malposition of the gastric banding, suggesting migration of the ring and total gastric outlet obstruction. She underwent two surgical procedures to relieve the obstruction.

Case 2

Patient 2 was born at the GA of 28 weeks. His mother, a 21-year-old primigravida had a history of gastric banding. Her nutritional status was never assessed. During pregnancy, she lost 23 kg of weight. After 25 weeks of gestation, there was excessive vomiting and diarrhoea. Foetal assessment by Doppler ultrasound at 28+6 weeks showed an end-diastolic block and haemodynamic signs of brain sparing. She was transferred to a tertiary care centre and the baby was born by emergency caesarean section. The Apgar score at 1 min was 2: no respiratory drive and bradycardia was present. The baby was intubated and ventilated.

The growth parameters of the neonate were weight of 1,250 g (P50), length 39 cm (P50–75) and head circumference 27.0 cm (P50).

Cranial ultrasound on the day of birth showed a bilateral grade III IVH (Fig. 1). Further evaluations showed progression of the cerebral and cerebellar bleeding.

Table 1 Overview of the described patients

	Case 1	Case 2	Case 3	Case 4	Case 5
Type of bariatric surgery	Gastric banding	Gastric banding	Gastric banding	Biliopancreatic diversion	Duodenal switch
Laboratory values mother	PT 46.8% (70–150%)	Pseudo-Bartter			Vitamin K 0.0008 nmol/L (0.8–5.3 nmol/L)
	aPTT 29.3 s (24–31 s)	K^+ 2.29 mmol/L (3.5–5.1 mmol/L)			
	f II 56% (70–130%)	HCO_3^- 29.7 mmol/L (22–29 mmol/L)			
	f V 121% (70–130%)				
	f VII 40% (70–130%)				
	f IX 75 % (70–130%)				
	f X 27% (70–130%)				
	Vitamin K1 0.2 nmol/L (0.8–5.3 nmol/L)				
Laboratory values infants	PT<10% (70–100%)	Pseudo-Bartter	PT 16.8% (70–100%)	PT 53% (70–100%)	
	aPTT 121.2 s (24–38 s)	K^+ 2.42 mmol/L (3.5–5.1 mmol/L)	aPTT 93.4 s (24–38 s)	aPTT 38 s (24–38 s)	
	Fibrinogen 1.29 g/L (2.00–3.80 g/L)		Fibrinogen 0.93 g/L (2.00–3.80 g/L)		
	f II 13% (70–130%)	HCO_3^- 27.8 mmol/L (22–29 mmol/L)	f II 18% (70–130%)		
	f V 78% (70–130%)		f V 50% (70–130%)		
	f VII 2.9% (70–130%)		f VII 2.6% (70–130%)		
	f IX 0.8% (70–130%)		f IX 8% (70–130%)		
	f X 4.2% (70–130%)		f X 13% (70–130%)		
Neonatal outcome	Died	Died	Mental retardation	Mental retardation	Died

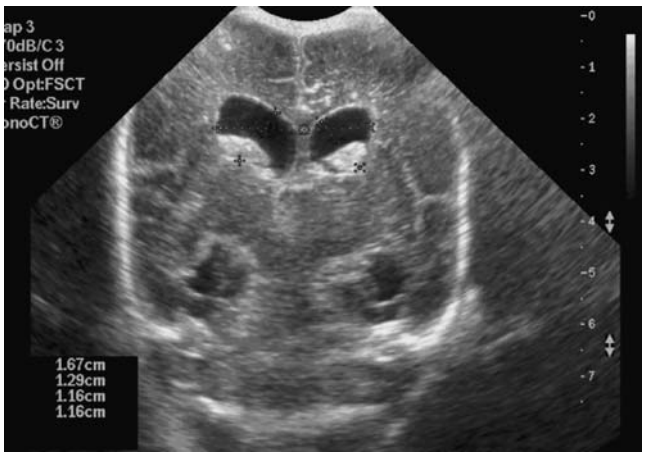

Fig. 1 Cerebral ultrasonography of patient 2 shows bilateral intraventricular bleeding with extravasation to the third and fourth ventricle and ventricular dilatation

Umbilical cord blood showed low haemoglobin of 13.7 g/dL (14.5–22.5 g/dL) and normal thrombocyte level: 194×10^9/L. There were a persistent hypokalaemia and alkalosis the first days of life (pseudo-Bartter) despite the severe bleeding (Table 1).

The neurological condition deteriorated progressively and after 7 days the newborn died.

The mother's biochemistry was severely disturbed with profound hypokalaemia (Table 1). She was treated with intravenous fluid and potassium supplements. Oesophago-gastroduodenoscopy showed dilatation of the cardia, suggesting gastric outlet obstruction and she had surgical removal of the gastric band.

After the surgical procedure, her nutritional status improved and the metabolic alkalosis and potassium normalised, thus proving she had no inherited Bartter syndrome.

Case 3

Patient 3 was born at the GA of 39 weeks. Her mother was gravida 2, para 1. The mother had a gastric banding in her

medical history. The pregnancy was 'uneventful' and the baby was delivered vaginally. However, with more detailed history taking, it was revealed that there was substantial maternal weight loss during pregnancy.

The neonate's growth parameters at birth were weight 3,265 g (P50–75) and the length 54 cm (P90). She was breastfed from birth, but unfortunately no information about vitamin K prophylaxis at birth was available, although this is a standard general practice.

At day3 of life, the neonate became extremely irritable. A lumbar puncture was done and yielded a bloody tap. A CT scan revealed a subdural haematoma. The neonate was then referred to a tertiary care centre for further management. The clotting profile was severely disturbed with very low values of all vitamin K-dependent factors (Table 1). Liver tests were normal. On admission (day3), she had mild thrombocytopenia: 94×10^9 with normal platelet function tests. Vitamin K and fresh frozen plasma were administered and the clotting profile normalised after day1. The neonate needed an urgent trepanation and developed a pitchy cry, nystagmus and convulsions in the postoperative period. Magnetic resonance scan (MR scan) showed bilateral infarctions and sequels of a subdural and subarachnoidal bleeding.

After a 7-year follow-up, she has a psychomotor delay and cerebral palsy.

Case 4

Patient 4 was born at GA of 40 weeks. Her mother was a primigravida with a history of biliopancreatic diversion. Pregnancy and delivery were uneventful. With further inquiry, it was determined that the mother had important weight loss during her pregnancy.

The neonate's birth weight was 2,455 g (<P3), length 47 cm (P5–10).

On day2, the baby presented with hypotonia, apneas, convulsions of the right arm and leg and nystagmus to the right side. The neonate was transferred to our tertiary centre for further management.

Cranial CT scan showed an extended subarachnoidal haemorrhage and cerebral oedema. There was a normal platelet count, but clotting profile showed a prolonged PT (Table 1). Currently, the child has a moderate mental disorder and follows special education.

Case 5

This case presented in Chicago University Hospitals.

A 27-year-old woman underwent a duodenal switch procedure and fell pregnant 1 year later. At that time, she had severe malnutrition. The 15 weeks' ultrasound scan was unremarkable. The 19 weeks' scan revealed a male foetus with shortened femurs and humeri bilaterally, nasal bridge hypoplasia, macroglossia, poorly defined hands and possible clubbed feet. Amniocentesis revealed a normal male karyotype. The lagging long bone measurements continued to worsen and ultimately the femurs were 6 weeks behind. The foetal thoracic circumference was two standard deviations below the mean, giving rise to concern for pulmonary hypoplasia. At 33+5 weeks, an emergency caesarean section was performed for foetal distress.

The birthweight was 1,468 g and Apgar scores were 1, 4 and 6 at 1, 5 and 10 min. Postnatal X-rays confirmed the antenatal ultrasound findings and demonstrated evidence of epiphyseal stippling. The neonate remained ventilated for 12 weeks when he died from respiratory complications due to pulmonary hypoplasia. Genetic testing did not identify any mutations associated with these dysmorphic features. The maternal vitamin K level was extremely low, 0.0008 nmol/L (normal range 0.8–5.3 nmol/L), and this may explain the skeletal malformations in this case.

Discussion

Bariatric surgery has become a cornerstone in the management of morbid obesity and is safely recommended to obese women of childbearing age [5]. The achievement of a pre-pregnancy weight reduction will increase fertility through reduction of hyperandrogenism and the incidence of polycystic ovarian syndrome. It will also decrease obesity-related risks for mother and foetus for e.g. pregnancy-induced hypertension, gestational diabetes, large-for-gestational-age infants, caesarean section and instrumental delivery [2, 6, 7, 13–15, 17, 18, 20–22, 26, 27].

Bariatric interventions can be classified in two types—malabsorptive interventions (performed by bypass surgery) or restrictive interventions.

Roux-en-Y gastric bypass and biliopancreatic diversion are two malabsorptive interventions. Neonatal complications caused by a deficiency of the maternal fat-soluble vitamins, in particular vitamin A, are well described [4, 12, 23], as well as foetal growth restriction [10, 13]. Maternal intestinal obstruction is also a reported complication. Symptoms may be misleading, as nausea and vomiting are also pregnancy-related complaints. However, this complication may lead to life-threatening conditions for mother and child [26]. Because of the possible risk of severe malnutrition, this type of interventions seems not appropriate for young fertile women.

In laparoscopic adjustable gastric banding (LAGB), an inflatable ring is placed around the gastric cardia. A small pouch is created, thus limiting food intake and adjusting the size of the remaining passage [20] and the gastric capacity of food intake. Band-related complications are rare,

including band leak and band migration. Deflation is needed frequently because of vomiting [26].

Pregnancies after restrictive bariatric interventions are generally considered to be safe [5, 6, 15, 21, 22, 26]; however, surgery-associated complications and severe nutrient deficiency can occur as a complication of this type of surgery as illustrated in our case series.

We are aware that not all cases are equally well documented, due to the fact that this is a retrospective report. But the similarity of the cases, together with the severe adverse outcomes of the neonates, makes it important to report.

In the first case, vitamin K deficiency causing disturbance of coagulation was well documented. The mother had a complete gastric outlet obstruction and secondary starvation with probable depletion of vitamin K stores. As there is a very limited placental transfer of vitamin K, any maternal vitamin K deficiency may provoke foetal vitamin K deficiency, increasing the risk of severe bleeding disorders [3, 9, 11, 19, 24].

Unfortunately, we had no coagulation test of the second patient, but the history and clinical symptoms are very similar to the first. We realise that this was a very preterm infant with an increased risk of intracranial haemorrhage due to prematurity, but the presentation with foetal end-diastolic block and brain sparing on sonar before delivery, poor Apgars, low haemoglobin at birth and intracranial bleeding stage III immediately after birth are all elements contributing to the hypothesis that there was already a foetal intracranial bleeding present.

The mother also developed a pseudo-Bartter syndrome as a result of chronic vomiting and so did the infant. Oesophagogastroduodenoscopy showed a gastric outlet obstruction, thus malnutrition with depletion of vitamin K stores is likely and therefore a similar pathogenesis is likely. The history and laboratory values of the third case are also very similar to the first patient.

Finally, vitamin K deficiency due to malabsorption is a plausible explanation of the pathogenesis of the haemorrhage of our fourth patient, but unfortunately the vitamin K-dependent clotting factors were not measured. Therefore, only the hypothesis that the haemorrhage was due to maternal vitamin K deficiency after bariatric surgery can be established, but with the maternal history it is of great importance and should be mentioned in this case.

Interestingly, in the last case, documented maternal vitamin K deficiency after bypass surgery resulted in a different manifestation with a rare and generalised bone malformation in the foetus. Rhizomelic chondrodysplasia punctata may be secondary to chromosomal anomalies, Mendelian gene disorders or teratogens like warfarin (vitamin K antagonist) [16]. Although no certainty can be obtained about a causal relationship, no other explanation for the skeletal dysplasia in this case could be found.

This dramatic series with poor outcome (three neonatal deaths and two severely disabled children) is very suggestive for the association between malabsorption and limited food intake due to excessive vomiting as a result of bariatric surgery or its complications and foetal and neonatal vitamin K deficiency resulting in severe intracranial haemorrhages and skeletal malformations similar to warfarin foetopathia, an anticoagulant working through vitamin K antagonism. As mentioned before, the weakness of our retrospective database is that not all cases are equally well documented, but the similarity of the cases, together with the severe adverse outcomes of the neonates, makes it important to report and as far as we know this is the first series of complications caused by vitamin K deficiency provoked by excessive vomiting or fat malabsorption after bariatric surgery.

Although there are major advantages of bariatric surgery before pregnancy in a certain patient population, it is not a single-time solution and one must consider pregnancies after bariatric surgery to be a risk [1, 8].

Nutritional care during pregnancy and prospective follow-up studies after bariatric surgery until some months after birth are needed to be able to detect the unusual complications and establish appropriate routine recommendations in these pregnancies [9, 26].

In summary, bariatric surgery in females of reproductive age may have some advantages, but the risk of malabsorption is real and it can have lethal foetal and neonatal consequences. These women need close follow-up by a multidisciplinary, specialised team before, during and after delivery to avoid severe malnutrition and vitamin depletion, especially for vitamin K.

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